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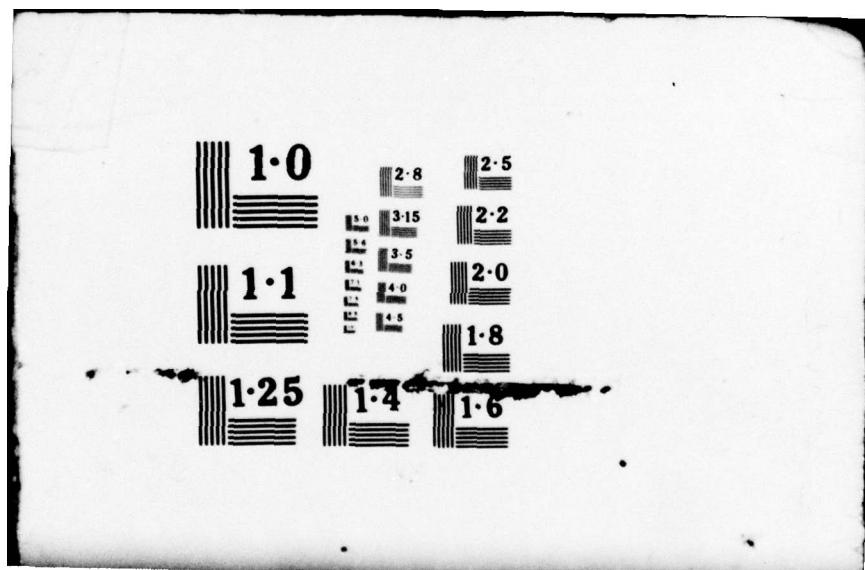
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NEW CONTROL ALGORITHMS VIA BRAIN THEORY*

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ABSTRACT

New concepts and viewpoints are needed to develop control algorithms for large scale systems. It is hypothesized that brain functions in human beings clearly demonstrate the existence of superior techniques for controlling complex systems with multiple objectives. The theory of compacta is investigated as a basis for learning, memory, and perception.

INTRODUCTION

In research areas like large scale systems and cybernetics, there is a great need for new concepts and viewpoints to handle effectively the complexity of the problems inherent in these systems. Classical control algorithms and design techniques are inadequate for these systems. The traditional and sound approach to developing a methodology for complex systems is to extend the tried and proven techniques which have been developed. The basic problem is that the procedure of extension from small to large exposes the system analyst to encountering many new and different problems from those encountered previously. That is, the system analyst can do a highly effective job on possibly an inadequately formulated problem.

An alternate approach to developing new control algorithms is to analyze highly complex biological systems that have evolved over time and exhibit some of the properties envisioned as needed for future generation large scale systems. In the field of cybernetics, the human being is often given as an excellent example of a highly complex system. The typical human being has a tremendous ability to learn, adapt, and perform and thereby reorder the environment in which he or she lives. This ability is derived from the capacity of the human brain to learn, recall, and innovate ideas, events, developments, etc. The close tie between brain functions and body muscular control is basic to the profound abilities of human beings.

A better understanding of brain function, which we call brain theory, would form the basis for a new and possibly different generation of control algorithms. We do not mean to draw an analogy between brain theory and digital computers. Yes, there are similarities between computers and brain function, but researchers in cybernetics-control theory have long recognized the inadequacy of this analogy. The idea that computers could be made to model brain function only if computers are made sufficiently fast, large in function and small in size has also been discredited. Of course future generation computer systems will execute tasks comparable to brain functions, but their organization, input and output patterns will most likely be totally different from any computer system now in existence or on the drawing boards. Some hard core problems need to be solved prior to formulating the architecture, language and programming of the needed computers. These hard core problem areas include input signal sampling techniques that are both spatial and temporal; information storage, modification, and retrieval; decision making procedures that involve both computational and logic tasks with variable algorithms to achieve non-unique solutions; command output signals that are both spatial and temporal; etc.

In this paper we seek to describe brain function in such a way as to account for memory and perception. Neurons in the human brain appear to function in large groups, called compacta. The neurons in such a group will reinforce each other's firing from moment to moment, so that the compactum as a unit can exist in a firing "state". The large number of elements in these groups make them very dependable in mental processes and suggests they may function as

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were the only kind of contacts to exist, this model would be worthless, for two reasons. First, weak contacts are fixed, in strength and in number, but in order for memory to occur, there must be some kind of physiological change occurring in the brain. Second, consider the difficulty of igniting a compactum by external stimuli (perceptions). A single neuron of the compactum requires impulses from at least 1 contacts in order to fire, but the probability of a given external simulation sending more than one impulse to the same neuron is, essentially, zero.

To circumvent these problems, Legendy proposed the existence of "latent" contacts and "strong" contacts. An impulse through a strong contact will cause a neuron to fire even if it receives no other impulses. A latent contact can become strong by frequent firings. Strong contacts are of two kinds. "Input" neurons have strong contacts on their dendritic ends, "output" neurons on their axonal ends.

The presence of strong contacts directly affects the two problems mentioned above. The problem of getting external stimuli to fire neurons is now solved, by assuming that external impulses impinge on a strong contact on an input neuron. Only one impulse is necessary to assure firing. The presence of strong and latent contacts also gives a mechanism for memory. For these contacts enable compacta to combine with other compacta to form "super" compacta, or "major" compacta. Thus, output neurons from several ignited "minor" compacta can send enough impulses to input neurons of other unignited compacta to cause a major compactum to fire. When contacts between two subcompacta are latent, and both subcompacta are firing, the continuous firing can cause the latent contacts to become strong. In this way, over a period of time, larger and larger compacta can be formed, representing more complexity in memory, or more "facts" in memory. By such a process of "facilitation" fewer and fewer minor compacta need be ignited to ignite the major compactum of which they are a part. Thus, if the minor compacta ignite in response to the perceptions of facets of an object, then the major compactum of which they are a part (whose ignition might correspond to the perception of the object "in toto") will occur with greater and greater frequency with the passing of time. Or, it will ignite when fewer and fewer "facets" are presented. This is the essence of recognition and, hence, of memory.

An interesting sideline to this theory concerns the presence of "images" in the brain. Consider that an external stimulus, such as a visual sensation, causes a major compactum to fire. This firing is the only record of the visual perception; that is, a particular compactum firing is associated with a corresponding visual image, and this firing is equated with the original stimulus. Now whatever makes this compactum ignite, its ignition will be equated again with the original visual stimulus. Another way of saying this is that the only thing ever "seen" is the ignition of certain compacta, so conversely whatever causes this ignition also causes "sight". This might suggest why memories are often so vivid as the realities they represent.

An extension of the preceding discussion leads to a model of perception.

A TENTATIVE MODEL OF PERCEPTION

Having seen how perceptions can cause compacta to fire, it is logical now to ask whether outside, random stimuli are the only source of compacta firing. In particular, consider the problem of internal stimulation of compacta. It is obvious that in any reasoning process, there is "something" in the psyche that can bring different "images" into play. If a person tells someone to concentrate on an orange, he can through some means bring the image of an orange to mind. An orange does not have to be present to make him "see" the image. One might object that the written or spoken word "orange" is necessary to bring the image to mind. To obviate the handling this objection, think of the simple case in which one is hungry. Suddenly, the image "orange" is perceived. There is obviously some feedback from the somatic state that influences the image which comes to mind. Or, continuing with the idea of compacta, there must be some process which depends on the somatic state that is capable of igniting the compactum or compacta corresponding to an orange.

Thus there are at least two types of phenomena which can excite compacta; exterior input and somatic, interior input. Now consider the more complex situation in which a combination of interior and exterior states excites compacta. The most obvious example is that of a thirsty man passing a water fountain in an unfamiliar environment. He sees the water fountain, and in it relief from thirst. The image comes to his mind of drinking from the fountain. The presence of the water fountain alone is not sufficient to make the image appear, for he may pass unfamiliar water fountains many times without noticing them. Likewise, his own somatic state is not necessarily responsible for the image, for he might have anticipated a different solution, such as going to a restaurant for a glass of water. Thus it must be the combination of states, interior somatic with exterior environmental, which causes the image.

This can be related to the idea of compacta in the following way; suppose the somatic state

the basic "building blocks" of memory and perception. When certain assumptions are made about the physical nature of neurons, compacta can be studied statistically. In particular, their existence and lower limit to their size can be established. A fairly simple stability problem can be solved using the mathematical formulae developed herein.

BACKGROUND

The science of mathematical neurophysiology has produced some very fine work in its short history. Perhaps the first study to suggest a workable scheme of the brain was the McCulloch-Pitts model of neural activity, propounded in 1943 in an article entitled "A Logical Calculus of the Ideas Immanent in Nervous Activity" [1]. This work described how the neurons of the cortex could be considered as logic units in a computing system, thereby giving a solid analytical basis to neural behavior. As important as this work was, it has become apparent that the brain differs greatly from the digital type of mechanism described there. The most obvious ways in which the brain diverges from a digital mechanism are its redundancy, its flexibility, and its "gestalt" recognition properties. It became very important to postulate a more "organic" approach to neural activity. In effect, having shown that a system such as the brain could function like a computer, and do the things that computers are able to do, there was a need to determine how the brain actually does work. To this end the work of C. R. Legendy [2], along with others, pointed out very fruitful directions. He suggested that the relevant "building blocks" of the brain are not the individual neurons, but are rather groups of neurons acting in concert. These groups and their behavior can be studied statistically in conjunction with some ideas of modern systems theory.

A DISCUSSION OF COMPACTA

According to Legendy, neurons in the cortex form groups, which he labelled "compacta" which have a "reverberatory" nature. This means that when each neuron of such a group is firing, each member continues to fire because of contacts with the other members of the group. Thus, the compactum will remain "ignited" until the neurons become "fatigued" or their thresholds become too high. Since the time required to fatigue the neurons is much longer than the duration of neuronal firing, these structures can be considered "stable." As a result, the firing of groups can be studied instead of the firing of individual neurons. The large numbers of neurons composing such groups facilitate statistical and probabilistic analysis which would be prohibitively difficult if applied to individual neurons.

Legendy suggested that these compacta could be ignited by perceptual impulses, and also that compacta could combine to form even larger groups, called "major compacta", by means of "strong" and "latent" contacts between neurons of different individual (or "minor") compacta. He then suggested how this model can account for several observed properties of memory, such as (1) the "gestalt" property of perception, whereby objects can be recognized even with some facets missing, (2) the very large memory capacity of the human brain, and (3) the observed lack of impairment of local and mental functions when whole areas of the cortex are removed.

Some of the difficulties of the Legendy model will be discussed, and a device which may extend the model from simply a memory scheme to an actual perceptual mechanism will be suggested. To facilitate this discussion it is necessary to formulate a simplified model of the neuron.

A MODEL OF THE NEURON

In order to proceed analytically, much of the fine structure of neurons must be ignored because of the mathematical difficulties involved. In particular, it is necessary to assume all neurons are similar, with the same number of dendrites and the same value of threshold potential τ . In addition, individual dendritic weights are all assumed to be unity, thus ignoring inhibition. Axonal lengths are assumed to vary in accordance with a distribution function to be discussed later.

The most drastic simplification is to discretize the time scale. All neurons are expected to act in synchronism with time increments Δt . Thus, if a neuron receives $\geq \tau$ impulses at time t_0 , it will fire at time $t_0 + \Delta t$ and simultaneously the neurons to which it sends contacts will receive these impulses. This assumption seems warranted when dealing with the neurons in a compactum because the compactum's firing, unlike that of an individual neuron, is a steady state process and thus the actual numbers of input impulses can be replaced by the average number of input impulses, at least for a first approximation. Of course, the steady state approximation breaks down after the neurons in a compactum become fatigued, but at this time the compactum simply stops firing as a result of increase of the individual threshold potentials. Thus fatigue acts as a natural delimiter of compactum firing.

The contacts which have been described here are known as "weak" contacts in Legendy's article. Their presence guarantees the existence of compacta, as will be shown later. If weak contacts

induces a certain number of input neurons to fire through some mechanism, such as the Reticular Activating System. Assume the exterior perceptions induce another number of input neurons to fire. Now suppose that the levels of these firing can be adjusted by some filtering mechanism, and that the resultant number of input neurons firing can be held more or less constant. In this way, the number of ignited minor compacta, and hence the number of ignited major compacta, can be held fairly constant, at such a level that the organism can adequately deal with the information content. This should produce a sort of movie effect, with the environment and the organism's homeostatic mechanism in the role of the projector, and the space of compacta as the screen.

Such a scheme may also throw some light on the associative process. For it is logical to assume that, as the exterior environment changes, some compacta may remain ignited as others stop firing. Assuming, again, that ignited subcompacta correspond to elemental aspects of observed phenomena, some continuity of mental imagery should be expected. This continuity is central to association in mental functioning.

Figure 1 gives an idea of how such a mechanism could work in conjunction with the organism's homeostatic mechanism to give a crude picture of the behavioristic process. In a very real sense, the process is the homeostatic mechanism. Notice that there is feedback from the approach and avoidance implementation schemes to the internal somatic states, meaning that once the major somatic needs of the organism are met, the homeostatic mechanism turns to needs of the next highest priority.

There is no need to assume that this simple on-off preference scheme is the only way the brain functions. If the theory of compacta is correct, probably the process is more like the following: the Reticular Activating System sets up a pattern of somatic "need" with internal stimuli feeding impulses into the cortex. As different external stimuli impinge upon consciousness, the internal stimuli act to reinforce the firing of compacta which are related to these needs. The brain, so to speak, ignites major compacta in the same way that a director screens actors for parts. Simuli from the internal and external states combine to ignite compacta which are tested for their suitability in the roles available.

The "play" in which all this takes place is the "need pattern" of the organism. This "need pattern" can be quite complex, and the more universal of these patterns become "myths", or archetypes. An example might be the need pattern, or archetype, associated with the concept of "Mother". Conversely, these archetypes owe their potency to the fact that they elucidate certain basic need states of the organism.

If the foregoing concepts are correct, it appears that the model proposed here may explain certain very basic mental functions. Thus a quantitative analysis seems merited. The beginning of such an analysis follows.

STEPS TOWARD A QUANTITATIVE MODEL

An important goal of this investigation is to prove the existence of compacta. Using initially the model of Legendy, it is considered that each neuron sends out n contacts, which are distributed with equal probability throughout the cortex.

This assumption does not conform very closely with the actual situation, but its similarity allows a straightforward mathematical analysis which can be adapted to a more complicated model later. There it will be shown that this simple model gives quite meaningful results.

Given a randomly chosen group of N neurons, consider the probability that this group comprises a compactum. For this to be true, each neuron in the group must receive contacts from at least r other neurons in the group. The probability of a given neuron having a contact from any other neuron is $\frac{n}{B}$, where B is the total number of neurons in the brain. The probability of having exactly m contacts from the $N-1$ other neurons is $\binom{N-1}{m} \left(\frac{n}{B}\right)^m \left(1-\frac{n}{B}\right)^{N-1-m}$. Since $\frac{n}{B} \ll 1$, it follows that for $m \ll N-1$, which is the case of interest, a Poisson distribution is described. The mean number of contacts will be $(N-1) \frac{n}{B} \approx \frac{Nn}{B}$ so that $\lambda = \frac{Nn}{B}$ and $p(m, \lambda) = \frac{e^{-\lambda} \lambda^m}{m!}$. To have a compactum it is required that every neuron receive r or more contacts. The probability of one neuron receiving this number of contacts is $p(r, \lambda) + p(r+1, \lambda) + p(r+2, \lambda) + \dots$. If this cumulative probability is represented as $f(r, \lambda)$, the probability of N random neurons being a compactum is $f(r, \lambda)^N$. But such a group can be

chosen in $\frac{B!}{(B-N)!N!}$ ways from the neurons. So the total expected number of compacta of size N will be:

$$E(N) = [f(\tau, \lambda)]^N \frac{B!}{(B-N)!N!} \quad (1)$$

Taking the natural logarithm and using the approximation $\ln X! \approx X \ln X - X$ gives the expression

$$\ln E(N) = N \ln f(\tau, \lambda) + B \ln B - (B-N) \ln(B-N) - N \ln N \quad (2)$$

For small N this expression is negative, representing essentially zero probability of the existence of even a single compactum of size N . The smallest compactum expected to exist will correspond to an N which causes $\ln E(N)$ to change from negative to positive.

Using $\lambda = \frac{nN}{B}$, $B = 10^{10}$, $n = 10^4$, $\tau = 100$, and a table of cumulative Poisson terms gives the following table:

N	λ	$f(100, \lambda)$	$\ln E(N)$
70×10^6	70	.00043037	-3.8×10^8
73×10^6	73	.00155738	-3.9555×10^7
74×10^6	74	.00234093	-1.2099×10^7
75×10^6	75	.00335244	1.48177×10^7
80×10^6	80	.017108	1.4098×10^7

Table I

It follows from this development that compacta may indeed exist, with the smallest compactum being of the order $N_0 = 75 \times 10^6$. Notice that the expected number of compacta of size N_0 is a very large number; $E(N_0) = \exp(1.48 \times 10^7)$. Offhand this result might appear disastrous to the theory, for it would indicate that any mechanism that ignites compacta by slowly raising the number of impulses to input neurons will have to be unreasonably sensitive to ignition to prevent neuronal firing from spreading throughout the cortex. A little consideration, however, shows that the counting scheme used here counts two compacta as different if even one neuron is different between them. In other words, the quantity $E(N)$ is much larger than the actual number of compacta present.

It is reasonable to ask how many of the $\frac{B!}{(B-N)!N!}$ groups enumerated can be counted as "different" compacta. A tentative answer might be that two compacta are "different" if the ignition of one does not appreciably enhance the chances of the ignition of the other. This problem is too complex to deal with here, and it shall simply be remarked in passing that its presence does not invalidate the approach outlined in the previous pages, since there the inquiry regarded only the existence of compacta and not their "individuality". More will be said concerning this later.

Having demonstrated the existence of compacta within the framework of Legendy's assumption of spatial homogeneity of contacts, one can embark with more confidence upon the beginnings of a more physiologically accurate model of compacta.

THE NO-TOUCH ASSUMPTION AND THE POISSON DISTRIBUTION

It is crucial to get some information on the sizes, shapes, densities and distributions of compacta in order to understand how to limit the number of them excited, since the homeostatic mechanism would avoid the dangerous situation inherent in the simultaneous firing of a high percentage of the total neuronal population. For a given spatial configuration in the cortical area, it is desirable to estimate the density of compacta having that configuration. In making this estimate assume that the density of contacts from a neuron is given by $\rho(r)$, where r is the distance between the neurons. The work will be much easier after making an additional assumption, which can be called the No-Touch Assumption; namely, given any two neurons, the a priori probability of a contact between them is much less than unity. This would appear to be incorrect with regard to very close neurons, but since almost all axons are noticeably longer than the dendrites, neurons in the close vicinity of each other are not apt to have contacts.

The advantage of the No-Touch Assumption is that the Poisson distribution can be used when

it applies. Consider the impulses being received through a particular neuron's dendrites from a particular group of m neurons located at a fixed distance away from the first. Assume a probability p of contacts between any one of the neurons and the one being considered. Then the probability of r contacts in all from the m neurons is $p^r(1-p)^{m-r} \frac{m!}{r!}$. In the case $p \ll 1$, $r \ll m$, this is a Poisson distribution;

$$d(r, \lambda) = \frac{e^{-\lambda} \lambda^r}{r!} \quad (3)$$

where $\lambda = mp$ is the mean.

The usefulness of the Poisson distribution is twofold. First, the single parameter λ gives all the information needed about the distribution and is simply the mean, an easily calculable entity. Secondly, the distribution of the sums of Poisson variables is also a Poisson distribution, with a λ that is simply the sum of the individual λ 's. This is most easily seen by considering the M.G.F. (Moment Generating Function) of a Poisson distribution, the term $e^{\lambda(e^t-1)}$. If two Poisson distributed variables are added, the resulting distribution will have an M.G.F. equal to the product of their two M.G.F.'s, or $e^{\lambda_1(e^t-1)} e^{\lambda_2(e^t-1)} = e^{(\lambda_1+\lambda_2)(e^t-1)}$. So the resultant distribution is simply

$$d(r, \lambda_1 + \lambda_2) = \frac{e^{-(\lambda_1+\lambda_2)} (\lambda_1+\lambda_2)^r}{r!}$$

Thus in looking at the distribution of contacts being sent from other neurons to a "test" neuron it is necessary only to find the mean number of contacts, and use that number as λ for a Poisson distribution.

THE CALCULATION OF λ

Again, consider a test neuron, imbedded with a Cartesian coordinate system, (X,Y,Z), at a point (x,y,z). It is necessary to find the mean number of contacts impinging on this neuron from all neurons in a certain group. Assume that

$$\rho(r) = \rho([(x-x')^2 + (y-y')^2 + (z-z')^2]^{\frac{1}{2}}) \quad (4)$$

is the probability of contacts from a neuron at (x,y,z) to (x',y',z') as a function of distance of separation. That is, if neuron A is separated from neuron B by a distance R, the probability of A sending a contact to B (or of B sending a contact to A) is $\rho(R)$. In addition, consider a group with spatial density $\sigma(x,y,z)$; thus in one unit of volume at point (x,y,z), there are $\sigma(x,y,z)$ neurons in this group. The expected number of contacts to a neuron at point (x,y,z) from all neurons in this group, which is designed by (x,y,z), is given by

$$(x,y,z) = \iiint_{-\infty}^{\infty} [(x-x')^2 + (y-y')^2 + (z-z')^2]^{\frac{1}{2}} \sigma(x',y',z') dx' dy' dz' \\ = \rho * \sigma \quad (5)$$

where it is understood that $\sigma(x',y',z') = 0$ if (x',y',z') is outside of the cortical area, and the symbol $*$ indicates convolution. Thus, the obvious thing to do is take the Fourier transform of both sides, giving

$$\phi(k_1, k_2, k_3) = P(k_1, k_2, k_3) \cdot \theta(k_1, k_2, k_3) \quad (6)$$

where ϕ , P , and θ are the Fourier transforms of ρ , σ , and σ respectively.

This equation gives the required function, in theory at least, when the inverse Fourier transform is taken. Alternatively, it is possible to work backwards, and inquire what spatial distribution will render a specified probability distribution, by rewriting the equation in the form

$$\theta(k_1, k_2, k_3) = \frac{\phi(k_1, k_2, k_3)}{P(k_1, k_2, k_3)} \quad (7)$$

This value of this formulation will become apparent shortly.

Having found (x, y, z) it is possible, theoretically, to calculate the number of compacta with spatial distribution $\sigma(x, y, z)$. Using the notation explained before for the cumulative Poisson distribution, and working with the natural logarithm of $E(N)$ gives

$$\begin{aligned} \ln E(N, \sigma) &= \iiint_{-\infty}^{\infty} [\ln f(\tau, (x, y, z))] \sigma(x, y, z) dx dy dz \\ &\quad + N \ln \sigma_0 - \iiint_{-\infty}^{\infty} \sigma(x, y, z) \ln \sigma(x, y, z) dx dy dz \end{aligned} \quad (8)$$

with $\iiint_{-\infty}^{\infty} \sigma(x, y, z) dx dy dz = N$ understood.

The first term comes about from taking the logarithm of $\prod_{i=1}^N f(\tau_i, (x_i, y_i, z_i))$ where the i

index runs over the positions of all the neurons. The second and third terms concern the number of ways that a group of size N can be chosen from a distribution σ , where the symbol σ_0 indicates the density of all neurons in the cortex. A neuron in position (x, y, z) can be replaced by any of $\frac{\sigma_0}{\sigma(x, y, z)}$ neurons and the distribution will be unchanged. Thus, the original probability must be multiplied by $\prod_{i=1}^N \frac{\sigma_0}{\sigma(x_i, y_i, z_i)}$. Taking the logarithm of this number gives the second and third terms in equation (8). The expression $\iiint_{-\infty}^{\infty} \ln \sigma dx dy dz$ is associated with the "information" (or the "entropy") of the distribution.

This form of the equation is usually intractable, given the lack of an analytical expression for $f(\tau, \lambda)$. But at least one case can be handled; that is, if σ is chosen such that δ is constant over the extent of the compacta. In this case the mean number of contacts received per neuron is constant over the volume of the compacta. Then $f(\tau, \lambda)$ will be the same for every neuron. Consider a spherical compactum with radius R_0 and λ specified to be λ_0 . Then

$$\delta(r) = \begin{cases} \lambda_0 & r \leq R_0 \\ 0 & r > R_0 \end{cases} \quad (9)$$

where r is measured from the center of the sphere. If the Fourier transform of δ is defined as

$$\phi(k_1, k_2, k_3) = \iiint_{-\infty}^{\infty} e^{i(k_1 x + k_2 y + k_3 z)} \delta(x, y, z) dx dy dz$$

then it can be shown that

$$\phi(k_1, k_2, k_3) = 4\pi\lambda_0 \left(\frac{\sin|k|R_0}{|k|^3} - \frac{R_0 \cos|k|R_0}{|k|^2} \right) \quad (10)$$

where $|k| = (k_1^2 + k_2^2 + k_3^2)^{\frac{1}{2}}$.

Now if analytical expressions for $p(x, y, z)$ and $P(k_1, k_2, k_3)$ can be found, equation (7) can be used to get $\theta(k_1, k_2, k_3)$ and then $\delta(x, y, z)$ and from this is calculated

$$N = \iiint_{-\infty}^{\infty} \delta(x, y, z) dx dy dz = \theta(0, 0, 0) \quad (11)$$

In this way it is possible to determine the functional dependence of $E(N)$ upon N , where it is understood that this particular probability is used. It is expected that $E(N)$ will be larger for this distribution than most others, because of the homogeneous distribution of probabilities.

This section is closed with a short discussion of a possible form of $\rho(r)$. It is desirable to find a form which is both tractable mathematically and accurate physiologically. A priori considerations indicate that the $\rho(r)$ dependence on r should roughly resemble Figure 2. This simply indicates that the probability of finding a contact between very close neurons or very distant neurons is small, and that the distribution has a rather broad maximum about the point r_0 , which shall be called loosely the "average" axonal length.

After much trial and error, it is found that possibly the most reasonable simple analytical expression for $\rho(r)$ is $\rho(r) = \beta r^2 e^{-\alpha r^2}$. This can be adjusted to have a maximum at $r = r_0$ by

manipulating α . β is adjusted so that $\int_0^\infty 4\pi r^2 \rho(r) dr = n$ where n is the total number of contacts per neuron. These two conditions can be expressed formally in terms of the equations

$\alpha = \frac{1}{r_0^2}$ and $\beta = \frac{2\alpha^{\frac{5}{2}}n}{3\pi^2}$. The values of r_0 and n can be determined experimentally. A sketch

of this function with $\alpha=1$, $\beta=1$ is shown in Figure 2b.

This is probably not the exact distribution function, of course. It is suggested because it is much more true-to-life than Legendy's model, and allows for axons which are very long (which, it is known, do exist) and also because of its simple form. Another nice feature of this function is the fact that its Fourier Transform can also be expressed analytically.

$$P(|k|) = \beta \left(\frac{\pi}{2}\right)^{\frac{3}{2}} e^{-\frac{|k|^2}{4\alpha}} \left[\frac{3}{2\alpha^2} - \frac{|k|^2}{4\alpha^3} \right] \quad (12)$$

This fact can be used, as shown previously, for calculating θ and thus $\delta(x,y,z)$.

STABILITY AND OTHER PROBLEMS

Perhaps the most important problem to emerge concerns the stability of compacta. Compacta have been shown to exist, but there is no guarantee that they can be ignited independently nor, indeed, that a few compacta may be ignited without producing the "snowball" effect of igniting the brain's neurons. In effect, the phenomena of compacta contains a wealth of crucial problems for stability analysis. This point is amplified in the mechanism of ignition itself. If compacta are ignited by impulses impinging on input neurons, it is necessary to ask whether only a few compacta can be ignited without igniting all, or a significant portion, of the brain's neurons.

Suppose a compactum is composed of N neurons scattered at random throughout the cortex. If σ_0 is the density of neurons in the cortex, then $\sigma = \frac{N}{B}\sigma_0$ is the density of compactum neurons in the cortex. Then for everywhere except close to the cortical surface, $\lambda = \frac{\sigma}{\sigma_0}n = \frac{Nn}{B}$ approximately, where λ is again the mean number of contacts to a test neuron in a compactum contributed by other compactum neurons. Here the only assumption made about the $\rho(r)$ function of the neuron is that the r_0 is not too large. The larger the r_0 , of course, the more substantial the dropoff of λ toward the surface of the cortex. Suppose, for a first approach, that λ is uniform over the cortical area. A more exact treatment, of course, would invoke the equations used toward the end of the previous section.

Then $\sigma = \frac{N}{B}\sigma_0$ is constant over the cortical area, and equation (8) yields

$$\begin{aligned} \ln E(N, \sigma) &= \iint_{-\infty}^{\infty} \ln f(\tau, \frac{Nn}{B}) \sigma dx dy dz + N \ln \sigma_0 - \iint_{-\infty}^{\infty} \sigma \ln \sigma dx dy dz \\ &= N \ln \left(\tau, \frac{Nn}{B}\right) + N \ln \sigma_0 - N \ln \sigma \end{aligned}$$

or $\ln E(N, \sigma) = N \ln \left(\tau, \frac{Nn}{B}\right) + N \ln B - N \ln N \quad (13)$

The reader should notice the similarity between this and equation (2), quite surprising in light of the simplicity of the earlier model. Indeed, they are so close that, for the solution of the equation for the smallest size of compactum expected, the solution for (2) may be substituted, giving again $N_0 = 75 \times 10^6$. The slight difference between the two equations probably arises from the specification of the spatially homogeneous nature of the present case.

For this special case some aspects of the stability problem may be discussed; in particular, the problem of extraneous ignition of compacta. Consider two compacta of size N_0 . $\frac{N_0^2}{B}$ neurons, on the average, can be expected to be shared by the two compacta. In addition, some neurons in the second compactum and not in the first will be ignited by the firing of the first. The probability of a given neuron being so excited is just $f(\tau, \lambda) = f(100, \frac{nN_0}{B})$ since no spatial homogeneity is assumed. Thus $N f(100, \frac{nN_0}{B}) + \frac{N_0^2}{B}$ neurons will be

"erroneously" excited. If $N_0 = 75 \times 10^6$, Table I shows that this number is about 80×10^4 . This number is only about 1% of the compactum size, and so there is negligible chance of one ignited compactum causing another to fire erroneously.

Admittedly, this case is oversimplified. Very seldom would only one or two compacta be expected to be firing. But the discussion above makes stability of compacta more believable, whereas a more sophisticated treatment is quite beyond the scope of this paper.

CONCLUSION

This is an introduction to, and certainly not an exhaustive treatment of, the theory of compacta. Indeed, only the basic concepts have been outlined, along with suggestions for formulating them mathematically.

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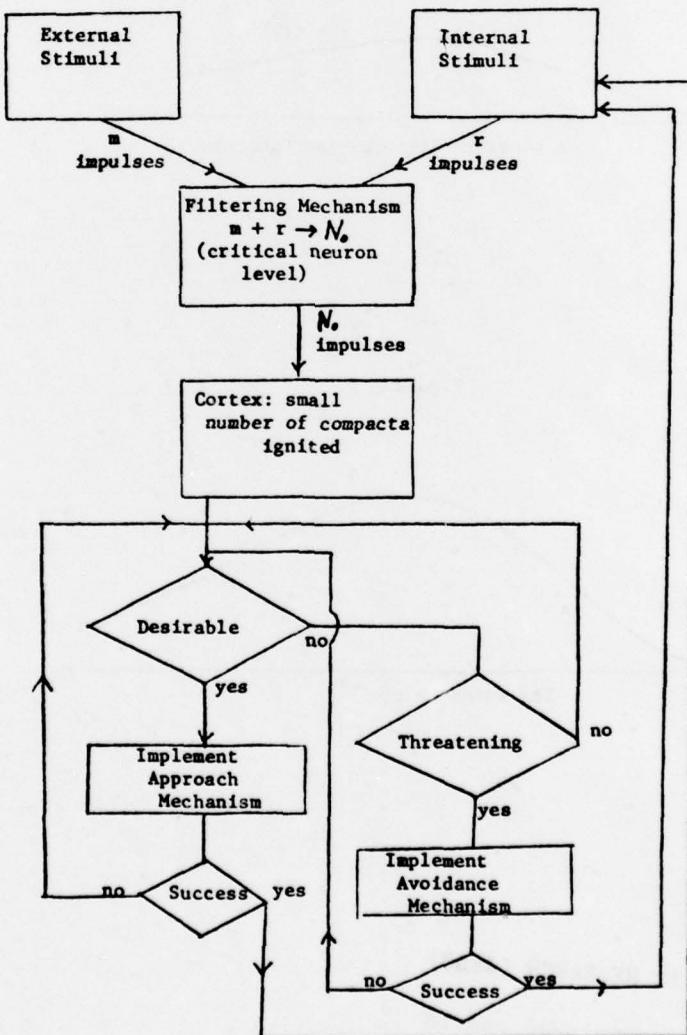


Figure 1. A Simple Behavioral Model

Figure 2a.

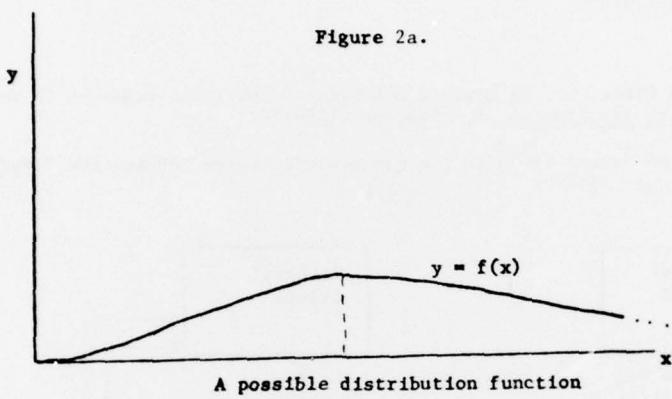
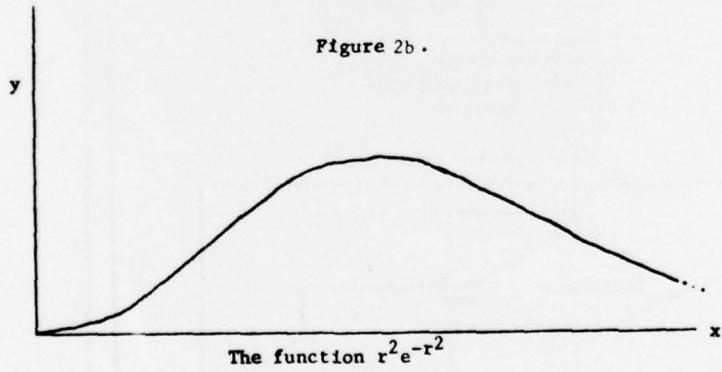


Figure 2b.



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